TABLE I. Patient Characteristics, Response to Therapy, and Survival

UPN	Age (yr)/sex	Duration of aplasia (mo)	IL-1α dose (μg/kg/d)	Immunosuppression	Initial response	Subsequent therapy	Survival (mo)
6382	14/F	2.4	0.1	ATG, MPRED	NR		>24.3
6850	74/M	1.3	0.1	ATG, MPRED	MR	Splenectomy	>22.4
7042	49/M	2.5	0.1	CSP, PRED	NR		22.7
7100	27/M	1.0	0.3	ATG, MPRED	NR	MM BMT	>29.8
7385	65/M	2.6	0.3	ATG, MPRED	MR	ATG, CSP, MPRED	>18.3
7392	30/M	1.8	0.3	ATG, MPRED	NR	URD BMT	>17.1

UPN = unique patient number; ATG = anti-thymocyte globulin; MPRED = methylprednisolone; CSP = cyclosporine; PRED = prednisone; NR = no response; MR = minimal response; MM BMT = mismatched related donor bone marrow transplant; URD = unrelated donor.

In summary, side effects of rhuIL-1α were well tolerated, but there was no demonstrable effect on hematopoiesis. In a previously reported study of four patients with severe idiopathic aplastic anemia who were refractory to immunosuppressive therapy, rhuIL-1α also did not effect significant hematologic improvement [4]. The time allowed for response in our study may have been too short to see an effect on marrow cellularity. The trial was terminated early when the manufacturer elected to discontinue development of this cytokine. Too few patients were studied to correlate response to rhuIL-1α to subsequent marrow recovery following immunosuppressive therapy.

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Abnormal Chromatin Clumping in Polymorphonuclears From a Patient With AIDS

To the Editor: Myelodysplastic changes are often detected in HIV-infected patients. To our knowledge, abnormal clumping of chromatin (ACC) in polymorphonuclears of HIV-infected patients has not been reported so far.

A 48-year-old HIV-infected homosexual man was found to be HIV+ in 1990. He remained asymptomatic until December 1994. He has been treated continuously with trimethoprim-sulfamethoxazole and successively with zidovudine and didanosine (discontinued in April 1993), and then with acyclovir and zalcitabine. He was admitted in December 1994 for a stage I B cell lymphoma of the ileum. He underwent complete surgical excision of the tumor and was discharged with trimethoprim-sulfamethoxazole therapy alone. On admission, hemoglobin was 163 g/l, platelets 216 × 10^9/L, and white blood cells (WBC) 5.9 × 10^9/l with 70% neutrophils, 4% immature granulocytes. Most granulocytes (70%) exhibited ACC associated with a monolobed nucleus resembling Pelger anomaly (Fig. 1). Granules were normal. Bone marrow (BM) was mildly hypocellular; ACC was striking in the granulocytes, faintly seen in some metamyelocytes, and absent from the other lines. Other laboratory data were: absolute CD4+ lymphocyte count, 20/mm^3; leukocyte alkaline phosphatase (LAP) score, 262 (normal range, 20–80); peroxydase activity, 382 (normal range, 20–80); nitroblue tetrazolium test, 76% (normal range, 75–80%); latex, 90% (normal range, >80%); the karyotype revealed no clonal anomaly; and the clonal growth of marrow progenitors in methyl-cellulose culture showed a poor colony-forming unit-granulocyte-monocyte (CFU-GM) growth: five colonies (normal range, 58–134), and eight clusters.

The morphological alterations: 1) were not seen in a previous blood smear carried out in our laboratory as the patient was undergoing zidovudine, zalcitabine, and trimethoprim-sulfamethoxazole therapy (April 1993); 2) persisted in March 1995, as no medication except trimethoprim-sulfamethoxazole had been administered for 3 months; and 3) have not been reported to occur with trimethoprim-sulfamethoxazole.

The granulocyte anomaly in our patient might be indicative of ACC syndrome. He presented with a normal blood cell count, few circulating immature cells, and mildly hypocellular BM, whereas most of the previously published cases of ACC syndrome showed frequent hyperleukocytosis, BM hypercellularity with granulocytic hyperplasia, and a high percentage of circulating myelocytes [1,2]. Such discrepancies may be due to: (1) the early stage of the disease; in a few previously reported cases, the leukocyte counts were normal at diagnosis and increased during the clinical course [2]; (2) the variability in the expression of the syndrome; the percentage of circulating immature cells and the leukocytosis have been reported with a wide incidence at the onset of the disease, ranging from 2–89%, and from 1.9 × 10^9/l–99.9 × 10^9/l, respectively [2,3]; and (3) the HIV infection: in our case, the very low CFU-GM growth could have been secondary to the HIV infection, according to the literature [4] .

Among the nucleus anomalies described in primary myelodysplasia, i.e., nuclear-cytoplasmic asynchronism, early clumping, pseudo-Pelger changes, moderate and sparse clumping chromatin, and ACC, all have been identified in HIV-infected patients with the exception of the latter [2,5]. Thus, ACC may merely be a new expression of HIV-related myelodysplasia.

Follow-up of our patient and further cases are needed to confirm the significance of these alterations.

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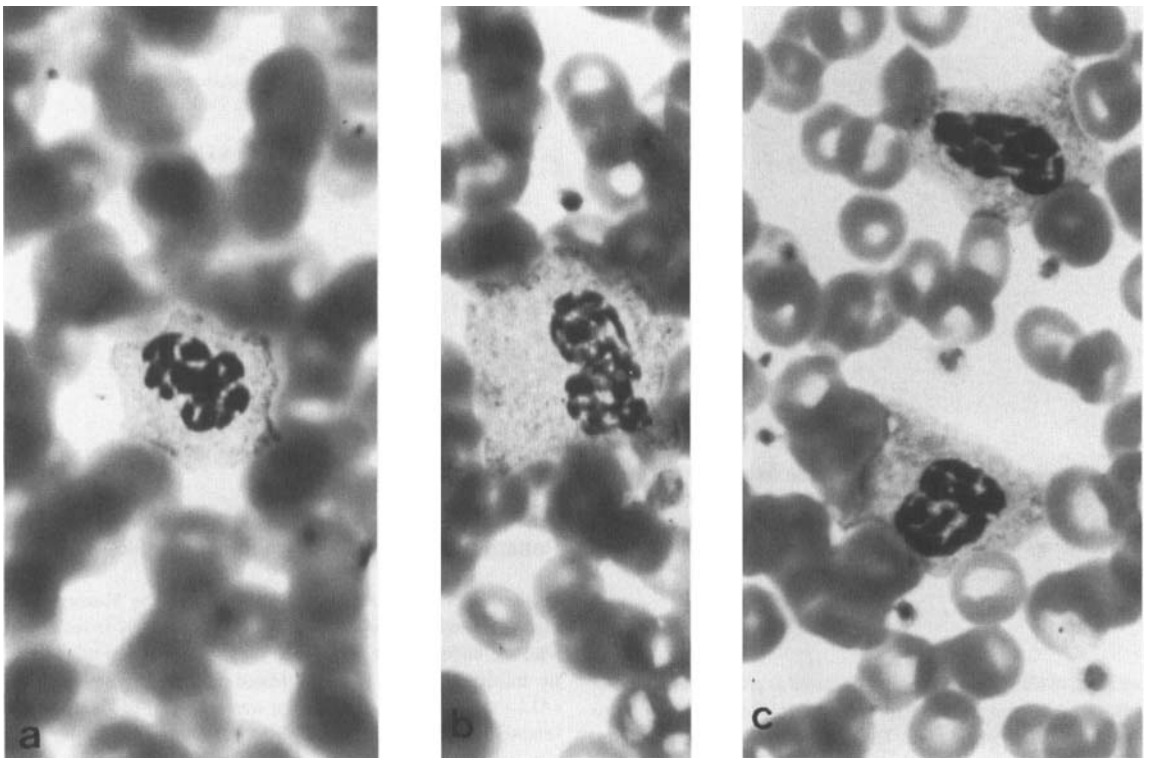

Fig. 1. Representative leukocytes from peripheral blood showing abnormal clumping chromatin. b: In a metamyelocyte. a and c: Associated with hyposegmentation in granulocytes. (May-Grünwald-Giemsa stain, ×1,200).

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Rearrangement of the *bcl*-6 Gene in Hodgkin's Disease, Lymphocyte Predominant Type

To the Editor: The *bcl*-6 gene encodes a protein belonging to the zinc-finger family, which regulates differentiation and development. A large study of 102 patients with large B-cell lymphoma revealed rearrangement of the *bcl*-6 gene in 20–25% of cases [1]. No such data exist for Hodgkin's disease. We report a case of Hodgkin's disease of the lymphocyte predominant type that was found to have a *bcl*-6 rearrangement.

A 53-year-old woman presented in January 1983 with enlarged lymph nodes over her left neck and a left breast mass. Biopsy studies of her lymph node and the left breast mass initially reported atypical lymphoid hyperplasia. Immunohistochemical study was inconclusive. She had persistent lymphadenopathy, and repeated lymph node biopsies in October 1983, October 1984, and August 1985 revealed a similar histology.

There was a progressive increase in the size of her left cervical lymph node by January 1987. Repeated lymph node biopsy revealed more definite features compatible with Hodgkin's disease, lymphocyte predominant, nodular type (LPHD). A review of all the biopsies showed a similar histological appearance, although the features were more typical in the later biopsies. The lymph nodes showed focal effacement of nodal architecture with replacement by nodular aggregates of lymphoid cells mainly composed of small lymphocytes; scattered L&H cells were identified. No typical Reed-Sternberg cells were found. The adjacent nodal tissue contained a few remnant follicles with some showing transformation of the germinal center. Immunohistochemical staining performed on paraffin sections showed that the L&H cells were positive for CD45 and the B-cell marker CD20 and negative for CD30, CD15, and the T-cell markers CD3 and CD45RO. The background small lymphocytes were composed of a mixture of B and T cells, with a predominance of the former. The features were in keeping with Hodgkin's disease, of the lymphocyte predominant, nodular subtype. Staging investigations found no evidence of disease dissemination. She received local radiotherapy, resulting in complete remission. She was last seen in March 1995, and there was no evidence of disease recurrence.

Frozen tissue obtained in 1987 was available for detection of *bcl*-6 gene rearrangement by Southern analysis. DNA was digested independently with *Bam*HI and *xba*I, and a 4-kb *Sac*-I-*Sac*-I fragment of the *bcl*-6 gene provided